In the specification:

Insert the paper copy of the Sequence Listing filed herewith following the Oath/Declaration.

Replace the paragraph beginning at page 13, line 3 with the following rewritten paragraph:

Therapy with antisense oligonucleotides is also intended to prevent the translation of proteins associated with a particular disease state. Osteopontin antisense molecules have been expressed by stably transfecting cells with a mammalian expression vector containing an osteopontin cDNA fragment in an inverted orientation. In that case, the antisense RNA was capable of targeting all forms of OPN mRNA, and expression in metastatic *ras*-transformed NIH3T3 mouse fibroblasts caused reduced malignancy. Primary tumor growth rates in nude mice and in a chick embryo assay for metastasis were reduced or completely inhibited (Behrend *et al.*, *Cancer Res.* 54: 832-837, 1994). Given the findings below, the present invention features methods for targeting OPN-b and OPN-c mRNA specifically. An antisense RNA, for example, that targets the exon 4/exon 6 splice junction will only inhibit translation of OPN-b, and an antisense RNA that targets the exon 3/exon 5 splice junction will only inhibit translation of OPN-c mRNA. In either of these cases, the full-length OPN-a continues to be translated. For example, the antisense oligonucleotide can be an RNA molecule (*e.g.*, an 18-mer, a 19-mer, a 20-mer, a 21-mer or a 30-mer), complementary to the region including and flanking the splice junction of OPN-b or OPN-c (*e.g.*, nucleotides 65-84, corresponding to OPN-b mRNA (SEQ ID NO:[2] 3) or nucleotides 84-103, corresponding to OPN-c mRNA (SEQ ID NO:[3] 5)).

Replace the paragraph beginning at page 22, line 25 with the following rewritten paragraph:

RNA was extracted from a variety of cell lines including the malignant cell lines MDA-MB-435, MDA-MB-231, 21MT1, 21MT2, Saos-2 and HeLa lymphoma cells; benign cell lines H16N2, MCF-7 and ZR75; the normal breast epithelial cell lines, 76N, 70N, 7VNE, 3VN and 7VN; breast epithelial cells immortalized with the HPV oncogene E6 including 81E6, M2E6E7 and 16E6P; and the T-cell line Jurkat. Osteopontin mRNA from several of these lines was analyzed by RT-PCR, cloning, and sequence analysis (see FIG 5). Primers for osteopontin amplified a 616 bp segment from the 5' end of the transcript. Reactions lacking a template and reactions in which GADPH was provided as the template served as controls. Alongside every other cell line, RNA from the malignant cell line MDA-MB435 cDNA was amplified to mark the two osteopontin bands that represent OPN-a and OPN-b. The two forms of osteopontin observed in the malignant cell lines MDA-MB435, HeLa, 21MT1, 21MT2 and Saos-2 were cloned and sequenced, and subsequently identified as OPN-a (SEQ ID NO:1; Fig. 1) and OPN-b (SEQ ID NO:[2]3; Fig. 2). In addition to the breast tumor cells described in the sequencing analyses (MDA-MB435, 21MT1 and 21MT2), gel mobility shift assays demonstrated that the breast tumor cell line MDA-MB-231 also expressed both OPN-a and the smaller variant OPN-b. Analysis by RT-PCR revealed the presence of various isoforms (FIG 6).